Candida pathogens induce protective mitochondria-associated type I interferon signalling and a damage-driven response in vaginal epithelial cells.

Pekmezovic M, Hovhannisyan H, Gresnigt MS, Iracane E, Oliveira-Pacheco J, Siscar-Lewin S, Seemann E, Qualmann B, Kalkreuter T, Müller S, Kamradt T, Mogavero S, Brunke S, Butler G, Gabaldón T, Hube B (2021) *Candida* pathogens induce protective mitochondria-associated type I interferon signalling and a damage-driven response in vaginal epithelial cells. *Nat Microbiol* 6(5), 643-657.

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Abstract

Vaginal candidiasis is an extremely common disease predominantly caused by four phylogenetically diverse species: Candida albicans; Candida glabrata; Candida parapsilosis; and Candida tropicalis. Using a time course infection model of vaginal epithelial cells and dual RNA sequencing, we show that these species exhibit distinct pathogenicity patterns, which are defined by highly species-specific transcriptional profiles during infection of vaginal epithelial cells. In contrast, host cells exhibit a homogeneous response to all species at the early stages of infection, which is characterized by sublethal mitochondrial signalling inducing a protective type I interferon response. At the later stages, the transcriptional response of the host diverges in a species-

dependent manner. This divergence is primarily driven by the extent of epithelial damage elicited by species-specific mechanisms, such as secretion of the toxin candidalysin by C. albicans. Our results uncover a dynamic, biphasic response of vaginal epithelial cells to Candida species, which is characterized by protective mitochondria-associated type I interferon signalling and a species-specific damage-driven response.

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